EXHIBIT A

Steven Noffsinger

Vs

The Valspar Corporation

Report of H. James Wedner, MD, FAAAAI

This report is based upon my review of the medical records and documents pertaining to Steven Noffsinger as well as deposition testimony of Mr. Noffsinger that I have been provided. It is also based upon the complaint of Mr. Noffisnger and the amended complaint, the deposition of Mr. Noffsinger, the medical records of The Defiance Clinic (which includes the majority of his medical records); the vocational assessment reports and the SSA reports and the report of Dr. Thomas H. Milby; as well as the accident investigation reports. It is my understanding that additional medical records and/or deposition testimony may become available and I will add to or amend this report, if necessary, once I have had the opportunity to review these documents.

I. I am a physician licensed to practice medicine in the state of Missouri. I am currently Professor of Medicine in the Department of Medicine at Washington University School of Medicine and Chief of the Division of Allergy and Clinical Immunology in the Department of Medicine. I also serve as the Director of the Training Program in Allergy and Immunology of the Washington University School of Medicine/Barnes-Jewish Hospital/Children's Hospital of St. Louis GME Consortium, which is supported by both the Department of Medicine and the Department of Pediatrics of Washington University Medical School. I also serve as the Medical Director of The Asthma and Allergy Center of Washington University School of Medicine. The Asthma and Allergy Center is a clinical setting located in suburban St. Louis County which specializes in the diagnosis and treatment of allergic and immunologic diseases including allergic reactions to airborne contamination; this includes the evaluation of patient with real or presumed exposure to high levels of pulmonary irritants. A copy of my current

curriculum vitae is appended to this document.

II. My duties as Professor of Medicine include: patient care; teaching of Fellows in allergy and immunology, medicine and pediatric residents and interns and medical students; and clinical research dealing with the exposure of individuals with asthma, allergic rhinitis and allergic skin disease to indoor and outdoor allergens with a concentration on allergenic fungi. As part of my clinical practice I am frequently called upon to evaluate and treat individuals who present with complaints of possible exposure upper and lower respiratory irritants or irritant containing products. At The Asthma and Allergy Center we have a full Pulmonary Function Laboratory where the formal evaluation of these patients can be performed. Because we are at a major medical center, Washington University School of Medicine, in many cases we see patients who have previously been evaluated in other centers throughout the mid-west or the country. There are many instances where we are called upon to confirm or deny the existence of reactive airways dysfunction syndrome or RADS

III. RADS -Reactive airways dysfunction syndrome, better known as RADS, was originally described by Dr. Stuart Brooks over 20 years ago. The characteristics of this disease where patients who had been previously healthy with no evidence of respiratory disease of other atopic condition who received an overwhelming exposure to a respiratory irritant. Each of the patients became ill within 24 hours of their exposure (most sooner) and presented to an emergency facility for treatment. Subsequent to this exposure and pulmonary reaction the patient would have a prolonged course that would be characterized by persistent abnormalities in pulmonary function that were similar if not identical to asthma accompanied by a clearly abnormal methacholine challenge test. Another characteristic of the original description of RADS was the absence of a history of allergies or positive skin tests. Subsequently there have been a number of modifications to the original Brooks criteria; the majority of these have been additions by Dr. Brooks himself. Dr. Brooks has noted that while

some of the patients recover, some do not. Some of the patients do have a history of allergies but none have had allergic asthma within the recent past; and finally some patients with childhood asthma who have been asthma free for some period of time, have been allowed into the RADS cohort. What remains clear is that the main characteristics of RADS (and/or related conditions that have been proposed see below) is that not respective of any antecedent conditions the patient must fulfill the following criteria: 1) there must have been an overwhelming exposure to an irritant sufficient to cause pulmonary injury and this must take the patient to the Emergency Department within 24 hours, 2) the patient must continue to have abnormal obstructive pulmonary function abnormalities and a positive methacholine challenge a 8 mg per milliliter or less, 3) there must be an absence of other conditions that could explain the patients condition.

IV. There have been a number of attempts to alter or expand the diagnosis of RADS to include a much broader spectrum of diseases. These include the so called irritant type asthma. This is a rather poorly defined group of conditions, which do not fulfill the formal criteria for RADS, but nonetheless some workers group as an irritant asthma RADS spectrum. A complete discussion of this topic is well beyond the scope of this report with the exception that not withstanding the designation, the patients with "irritant asthma", must still fulfill the criteria for asthma. That is, one can ignore causation until the diagnosis of asthma has been established. If the patient does not have an obstructive picture on pulmonary function testing, does not have 12% reversibility with a short-acting bronchodilator (albuterol) and/or does not have a positive methacholine challenge then that patient does not have asthma and does not have RADS or irritant asthma for that matter. An irritant exposure may have been noted, but in the absence of positive testing results the diagnosis cannot be RADS.

V. It should be noted that symptomatology cannot be used as the only criteria for a diagnosis of asthma. There are a number of conditions, physiological,

psychological or a combination of the two that can lead to symptoms that are mistaken for asthma. Indeed there are some situations in which patients present to our clinic with a diagnosis of severe persistent asthma where we are able to easily demonstrate that the patient does not have asthma and to find an alternative explanation for their symptoms. In some cases the patient may suffer from vocal chord dysfunction or VCD. It is not always possible to make the diagnosis of this condition by a simple nasopharyngoscopy or even while the patient is riding on a stationary bike. In our clinic, for example, we generally perform an exercise challenge followed by the rhinoscopy to observe the vocal chords when the patient has complained of shortness of breath and fatigue and this will bring out the abnormal movement of the chords that were not seen when the patient was in the resting state or even actively exercising Another condition that has recently come to the front is tracheo-bronchiomalacia. This is a condition that can only be diagnosed using a high resolution CT scan in full inspiration and full expiration. This has not been an exhaustive discussion of those conditions that mimic severe asthma, rather it is included to indicate that there are many and the physician should remember that when the PFTs do not match the symptoms, then an alternative explanation should be sought.

VI. With this discussion in mind, let us turn to Mr. Noffsinger. The fact of Mr. Noffsinger's exposure and his subsequent medical treatment are well documented in his medical records and will not be reiterated here. However, the question is, does Mr. Noffsinger fulfill the criteria for RADS or a RADS related condition. Mr. Noffsinger stated that he was exposed to paint fumes while sleeping in his truck cab; however, he did not present to an emergency facility within the 24-hour time point that is generally considered to be a hallmark for the onset of RADS. Indeed he did not seek medical attention for over a month (February 17, 2007 to March 20, 2007) after his exposure. This is inconsistent with any exposure related asthma that has been described in the literature. More importantly the initial diagnosis was not asthma it was pneumonia.

VII. Without any further exposure to any irritant or toxicant Mr. Noffsinger began to experience continued symptoms including cough which was persistent, burning in his chest and breathlessness along with shortness of breath with exertion. He was treated symptomatically by a number of his physicians with medications including inhaled corticosteroids and long-acting beta agonists (alone and in combination) and leukotriene modifiers. He has used high levels of short-acting bronchodilators (albuterol) both by metered dose inhaler and by nebulization. None of these medications have made a significant difference in his lung symptoms. He has also been treated with oral corticosteroids, again with little or no improvement.

VIII. Mr. Noffsinger has been evaluated by a number of pulmonary function laboratories both at the Defiance Clinic and at Ohio State School of Medicine. The results have been quite consistent. Routine PFTs have been within the normal range on multiple occasions, and have not provided evidence consistent with a diagnosis of asthma or RADS. That is, his flows have not demonstrated an obstructive pattern. This is best demonstrated by a normal FEV1/FVC and just as significant is the lack of response to an aerosolized bronchodilator. While there has been at time a slight decrease in the FVC and the slow FVC (a better measure of total lung capacity) this is more consistent with a restrictive picture than an obstructive one.

Mr. Noffsinger underwent a methacholone challenge on January 8, 2009, which was negative. He never crossed the 20% fall in FEV1 even at the highest concentration of methacholine (25mg/ml) that was used. For a test to be positive, the PC20 or the concentration that produces a fall in FEV1 of 20% or greater must be at or below 8 mg/ml. This clearly demonstrated that Mr. Noffsinger does not have reactive lung disease of any kind; i.e. RADS, so-called irritant-induced asthma or even intrinsic or extrinsic asthma. Mr. Noffsinger also underwent an exercise challenge, which demonstrated that he was able to maintain his blood oxygen within the normal range, demonstrating that he has

no deficit in his lung parenchyma.

Finally, neither Mr.Noffsinger's chest x-rays nor his pulmonary function testing has demonstrated significant air-trapping. A patient with severe refractory asthma from any cause would be expected to have some evidence of air-trapping confirming that Mr. Noffsinger does not suffer from this condition. Mr. Noffsinger has been evaluated for vocal chord dysfunction on several occasions and his chords have been noted to move properly and to be normal in character. However, as noted above he has had rhinoscopy during exercise, (in my experience we often see the change in vocal chord function after the patient has finished exercising and is catching their breath), which to my mind does not eliminate this diagnosis as the cause or a partial cause of this gentleman problem.

IX. Dr. Milby has opined the Mr. Noffsinger's methacholine challenge test is flawed and his use of albuterol several hours prior to the test is responsible for his lack of response to methacholine. Several points are pertinent. First, the half-life of albuterol is between 2 and 3 hours. Thus, even in an individual who had not been using this drug frequently, the PC20 curve may have been shifted a bit to the right by one dose but a single dose of albuterol several hours prior to the test would not have changed a positive test to a negative one. This would be true particularly for a patient who has very severe asthma as has been suggested for Mr. Noffsinger. More importantly, Mr. Noffsinger is a very frequent user of short-acting bronchodilators and patient who over-uses these drugs have their beta-receptors down regulated. That is, the effect of the drug is markedly diminished. Indeed, that is the reason they take more and more of the drug with less and less effect. What this means is that the dose of albuterol that Mr. Noffsinger took prior to his challenge test was inconsequential and had no effect on his methacholine challenge. Thus, the challenge was a valid test demonstrating the Mr. Noffsinger did not have bronchial hyper-reactivity.

The second problem that is noted by Dr. Milby, progressive fatigue due to repeated testing is a well-known phenomenon and is actually taken into account in the interpretation of the test. Note that the testing looks at FEV1 only and not FEV1/FVC. Because of the way the test is performed, fatigue will actually make a test more positive and not turn a positive test negative. In point of fact, Mr. Noffsinger did have a modest decrease in FVC (from 4.06to 3.39) a 16.5% change but his FEV1 never went below -14.4% at 25 mg/ml. Thus, although a bit tired, Mr. Noffsinger still had a normal test.

x. What then is Mr. Noffsinger's current pulmonary condition? Mr. Noffsinger has been evaluated on multiple occasions and does not fulfill the criteria for asthma, irritant-induced asthma (if this condition exists) or RADS. Indeed, he does not have any abnormality that is consistent with an obstructive pulmonary defect. In addition, he does not have any other ventilatory defect and is able to appropriately oxygenate his blood during exercise. Mr. Noffisnger's symptoms are clearly inconsistent with his measured pulmonary function and his chest x-ray. In cases such as Mr. Noffisnger's several diagnoses come to mind. The first is VCD. While Mr. Noffsinger has had several rhinoscopies, one on an exercise bike, with negative results, in our experience this does not rule out this diagnosis and an exercise challenge followed by rhinoscopy should be considered. Second, tracheo-broncheomalacia should be considered. While the, "birds-beak", shape of the flow-volume loop is not present, this is not an obligatory finding, and a high-resolution CT of the chest in full inspiration and full expiration is warranted. Neither of these conditions is the result of or even related to any presumed exposure to paint fumes, however.

Finally, it should be pointed out that there are patients who have no true pulmonary abnormality and simply believe that they have asthma. These patients have all of the symptoms of this disease, but when evaluated with the appropriate testing have no physiologic abnormality. They take increasing amounts of medication and do not improve. The cause of this condition is

unknown. Again, however, this condition is not exposure related.

XI. Conclusions. To a reasonable degree of medical and scientific certainty, Mr. Noffsinger does not suffer from RADS or any other type of reactive lung disease including bronchial asthma. He does not have work-related, occupational or irritant-induce asthma as has been suggested by Dr. Milby. He did not suffer any significant or long lasting adverse health effects from any exposures to that might have occurred on February 17, 2007.

H. James Wedner, M.D., FAAAAI